QT dispersion: Does it worsen with the increasing number of affected coronary vessels?

QT dispersiyonu: Koroner damar tutulumu arttıkça daha da mı kötüleşir?

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There is a considerable evidence that QT dispersion (QTD) may indirectly reflect underlying non-homogeneity of ventricular repolarization (1). An increase in QTD is reported to predict the occurrence of life-threatening ventricular tachyarrhythmias and sudden cardiac death in patients with ischemic heart disease (2). Furthermore, QTD was found to increase during episodes of myocardial ischemia or infarction (3). In a cross-sectional study design, we tried to explore a possible relationship between QTD and the number of affected coronary vessels in a series of patients admitted for elective coronary angiography.

We enrolled 60 consecutive patients admitted to our catheterization laboratory for elective coronary angiography, during the period from January to June 2005. We included only patients with significant stenosis of at least one epicardial coronary artery as explained afterwards. We excluded patients with conditions likely to prolong QT interval: recent myocardial infarction or cerebrovascular stroke in the preceding 4 weeks, and patients taking medications known to prolong the QT interval - quinidine, amiodarone, etc. Before inclusion, an informed consent was obtained and the study protocol was approved by our Institutional Human Research Committee.

All patients underwent selective left and right coronary arteriography using the standard technique and the angiographic data were individually analyzed by an independent expert interventionalist, blinded to the electrocardiographic findings. Significant coronary stenosis (assessed by visual estimation) was defined as 70% or more luminal obstruction of at least 1 sizable epicardial coronary artery, seen in 2 different projections or at least 50% luminal obstruction of the left main coronary artery. Patients with significant stenosis of the left main coronary artery were considered to have double-vessel disease and those with significant stenosis of the left main coronary artery and right coronary artery were considered to have three-vessel disease. Patients with previous coronary artery bypass surgery were assessed regarding the patency of the grafts and the non-grafted sizable native vessels. Based on the above definitions, we enrolled 20 patients with single-vessel disease (SVD group), 20 patients with double-vessel disease (DVD group) and 20 patients with three-vessel disease (TVD group).

All included patients underwent a resting high-quality 12-lead electrocardiographic recording, which was subsequently evaluated by an expert electrophysiologist blinded to the clinical and angiographic data. QT interval was measured with the manual technique, as the time in milliseconds (msec) between the first deflection of the QRS complex and the point of return of the T wave to the isoelectric line. We averaged three consecutive complexes in each lead. We recorded the maximal and minimal QT intervals, and calculated the QTD as the difference between both intervals, recorded individually for each patient. We calculated then the corrected QT (QTc) interval from the Bazett's formula as follows: QTc interval=QT interval / $\sqrt{RR}$ (4). Finally the QTc dispersion (QTcD) was calculated as the difference between the maximal and minimal QTc intervals. The mean values of QT and QTc intervals were calculated for each group separately, as well as the mean values of QTD and QTcD.

The mean age of the whole study cohort was 54.4±13 years, 54 (90%) being males. The three individual groups were matched regarding age, sex, smoking, resting heart rate, and prior coronary revascularization. However, patients in the TVD and DVD groups were more likely diabetic versus the SVD group ($p<0.05$). Moreover, TVD patients were more likely hypertensive versus the DVD and SVD groups ($p <0.001$).

We found that both QTD and QTcD increased significantly with the increasing number of stenotic coronary vessels (48±16, 80±18 and 120±34 msec; and 53±19, 89±21 and 142±19 msec, for SVD, DVD and TVD, respectively, $p<0.0001$ for both) (Table 1).

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Multivariate regression analyses identified diastolic blood pressure, the presence of two-vessel and three-vessel disease by coronary angiography, as independent predictors of increased QTD, while identified prior coronary bypass surgery, the presence of two-vessel and three-vessel disease by coronary angiography, as independent predictors of increased QTcD (Table 2).

It is hypothesized that myocardial ischemia alters the repolarization properties of cardiac myocytes, creating a difference in action potential duration between ischemic and non-ischemic regions of the ventricular myocardium (5). It is assumed that with increasing number of stenosed coronary arteries, a wider area of myocardial ischemia exists, with less likelihood of ischemic regions being supplied by collateral circulation, and hence a greater propensity to non-homogeneity of ventricular electrical recovery among adjacent segments of the ventricular myocardium. This would create a potential for re-entry circuits, and set the stage for ventricular tachyarrhythmias. It was formerly shown that patients with three-vessel coronary artery disease and preserved systolic function have a higher mortality at 5 years follow-up as compared to those with single-vessel disease (6). The higher QTD in three-vessel disease patients, demonstrated in our series may well contribute to this worse outcome.

Previously, Yılmaz et al. (7) reported a significant increase in QTcD between patients with significant (more than 50%) stenosis versus those with normal or insignificant stenosis. Yet, our study was the first to demonstrate that a gradient of progression in QTD also exited with the increasing number of affected vessels. Stierle et al. (5) found that QTD increased significantly following incremental atrial pacing, and corresponded with the extent of ischemia as reflected by cardiac lactate extraction ratio. However, they conducted their work on a cohort of three vessel disease patients without comparison to other patients with less extensive coronary involvement.

Table 1. QT dispersion and QT intervals (maximum and minimum) in the study groups

Variables	SVD Group (n=20)	DVD Group (n=20)	TVD Group (n=20)	p
QTD, msec	48±16	80±18	120±34	<0.0001
QTcD, msec	53±19	89±21	142±19	<0.0001
QT max, msec	396±22	416±23	454±41	<0.0001
QTc max, msec	448±36	460±49	507±42	<0.0001
QT min, msec	348±26	336±23	328±33	>0.05
QTc min, msec	389±25	376±38	371±35	>0.05

All variables are presented as mean±SD
DVD - double- vessel disease, max - maximal, min - minimal, msec - milliseconds, QTc - corrected QT interval, QTcD - corrected QT dispersion, QTD - QT dispersion, SVD - single- vessel disease, TVD - three- vessel disease

Table 2. Multivariate linear regression model demonstrating the independent predictors of QT dispersion

Variables	β Coefficient	p
Male gender	0.010	0.914
Diabetes	0.104	0.205
Hypertension	-0.130	0.362
Smoking	0.080	0.377
Prior percutaneous coronary intervention	0.069	0.388
Prior coronary bypass surgery	0.027	0.722
Heart rate	-0.012	0.897
Systolic blood pressure	0.073	0.625
Diastolic blood pressure	0.268	0.039
Two-vessel disease	0.258	0.006
Three-vessel disease	0.770	0.000

Conflict of interest: None declared

References

1. Roukema G, Jagmeet P Singh, Meijs M, Carvalho C, Hart G. Effect of exercise-induced ischemia on QT interval dispersion. Am Heart J 1998; 135: 88-92.
2. Kramer B, Brill M, Bruhn A, Kubler W. Relation between the degree of coronary artery disease and left ventricular function and the duration of the QT-interval in ECG. Eur Heart J 1986; 7: 14-24.
3. Tıkız H, Terzi T, Balbay Y, Demir AD, Soylu M, Keleş T, et al. QT dispersion in single coronary artery disease: is there a relation between QT dispersion and diseased coronary artery or lesion localization? Angiology 2001; 52: 43-51.
4. Bazett HC. An analysis of the time relations of electrocardiograms. Heart 1920; 11: 353-70.
5. Stierle U, Giannitsis E, Sheikhjdeh A, Krüger D, Schmücker G, Mitusch R, et al. Relation between QT dispersion and the extent of myocardial ischemia in patients with three vessel coronary artery disease. Am J Cardiol 1998; 81: 564-8.
6. Lopes NH, Paulitsch Fda S, Gois AF, Pereira AC, Stolf NA, Dallan LO, et al. Impact of number of vessels disease on outcome of patients with stable coronary artery disease: Five-year follow-up of the Medical, Angioplasty and bypass Surgery study (MASS). Eur J Cardiothorac Surg 2008; 34: 702-3.
7. Yılmaz R, Demirbağ R, Gür M. The association of QT dispersion and QT dispersion ratio with extent and severity of coronary artery disease. Ann Noninv Electrocardiol 2006; 11: 43-51.